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NEWS 5	FEB 06	Patent sequence location (PSL) data added to USGENE
NEWS 6	FEB 10	COMPENDEX reloaded and enhanced
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NEWS 10	FEB 23	Several formats for image display and print options discontinued in USPATFULL and USPAT2
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NEWS 20	MAR 30	IMSPATENTS reloaded and enhanced
NEWS 21	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS 22	APR 07	STN is raising the limits on saved answers
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NEWS 24	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS 25	APR 28	CAS patent authority coverage expanded
NEWS 26	APR 28	ENCOMPLIT/ENCOMPLIT2 search fields enhanced
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=> s TMC278
L1 19 TMC278

=> s l1 and tenofovir
1232 TENOFOVIR
L2 2 L1 AND TENOFOVIR

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=> s tenofovir
L3        1232 TENOFOVIR

=> s l3 and NNRTI
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          1257 NNRTI
                 (NNRTI OR NNRTIS)
L4         103 L3 AND NNRTI

=> s L4 and emtricitabine
           542 EMTRICITABINE
L5          42 L4 AND EMTRICITABINE

=> s l5 and compositions
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             2 COMPOSITIONSES
        345674 COMPOSITIONS
                 (COMPOSITIONS OR COMPOSITIONSES)
        648049 COMPNS
        744805 COMPOSITIONS
                 (COMPOSITIONS OR COMPNS)
L6           0 L5 AND COMPOSITIONS

=> s l5 and HIV
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           111 HIVS
         84600 HIV
                 (HIV OR HIVS)
L7          41 L5 AND HIV

=> s l7 and py <2003
      22984051 PY <2003
L8           7 L7 AND PY <2003

=> d l8 1-7 ibib ab

L8   ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:        2004:403774  CAPLUS
DOCUMENT NUMBER:         141:374319
TITLE:                   Molecular targets and compounds for anti-HIV
                         therapy
AUTHOR(S):               De Clercq, E.
CORPORATE SOURCE:        Rega Institute for Medical Research, Katholieke
                         Universiteit Leuven, Louvain, B-3000, Belg.
SOURCE:                  Biomedical and Health Research (2002),
                         55(Drug Discovery and Design), 272-278
                         CODEN: BIHREN; ISSN: 0929-6743
PUBLISHER:               IOS Press
DOCUMENT TYPE:           Journal; General Review
LANGUAGE:                English
AB   A review.  Virtually all the compds. that are currently used, or under
     advanced clin. trial, for the treatment of HIV infections,
     belong to one of the following classes: (i) nucleoside/nucleotide reverse
     transcriptase inhibitors (NRTIs): i.e., zidovudine (AZT), didanosine
     (ddI), zalcitabine (ddC), stavudine (d4T), lamivudine (3TC), abacavir
     (ABC), emtricitabine [(-)FTC], tenofovir (PMPA)
     disoproxil fumarate; (ii) non-nucleoside reverse transcriptase inhibitors
     (NNRTIs): i.e., nevirapine, delavirdine, efavirenz, emivirine
     (MKC-442); and (iii) protease inhibitors (PIs): i.e., saquinavir,
```

ritonavir, indinavir, nelfinavir, amprenavir, and lopinavir. In addition to the reverse transcriptase and protease step, various other events in the HIV replicative cycle are potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120 (polysulfates, polysulfonates, polyoxometalates, zintevir, neg. charged albumins, cosalane analogs); (ii) viral entry, through blockade of the viral coreceptors CXCR4 and CCR5 [bicyclams (i.e. AMD3100), polyphemusins (T22), TAK-779, MIP-1α LD78β isoform]; (iii) virus-cell fusion, through binding to the viral glycoprotein gp41 [T-20 (DP-178), T-1249 (DP-107), siamycins, betulinic acid derivs.]; (iv) viral assembly and disassembly, through NCp7 zinc finger-targeted agents [2,2'-dithiobisbenzamides (DIBAs), azadicarbonamide (ADA) and NCp7 peptide mimics]; (v) proviral DNA integration, through integrase inhibitors such as L-chicoric acid and diketo acids (i.e. L-731,988); (vi) viral mRNA transcription, through inhibitors of the transcription (transactivation) process (fluoroquinolone K-12, Streptomyces product EM2487, temacrazine, CGP64222). Also, in recent,years new NRTIs, NNRTIs and PIs have been developed that possess resp. improved metabolic characteristics (i.e. phosphoramidate and cyclosaligenyl pronucleotides of d4T), or increased activity against NNRTI-resistant HIV strains [second generation NNRTIs, such as capravirine and the novel quinoxaline, quinazolinone, Ph Et thiazoly-lthiourea (PETT) and emivirine (MKC-442) analogs], or, as in the case of PIs, a different, non-peptidic scaffold [i.e. cyclic urea (DMP 450), 4-hydroxy-2-pyrone (tipranavir)]. Given the multitude of mol. targets with which anti-HIV agents can interact, one should be cautious in extrapolating from cell-free enzymic assays to the mode of action of these agents in intact cells. A number of compds. (i.e. zintevir and L-chicoric acid, on the one hand, and CGP64222 on the other hand) have recently been found to interact with virus-cell binding and viral entry in contrast to their proposed modes of action targeted at the integrase and transactivation process, resp.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:891974 CAPLUS

DOCUMENT NUMBER: 139:46106

TITLE: New anti-HIV agents and targets

AUTHOR(S): De Clercq, Erik

CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.

SOURCE: Medicinal Research Reviews (2002), 22(6), 531-565
CODEN: MRREDD; ISSN: 0198-6325

PUBLISHER: John Wiley & Sons, Inc.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Virtually all the compds. that are currently used or are subject of advanced clin. trials for the treatment of HIV infections, belong to one of the following classes: (i) nucleoside reverse transcriptase inhibitors (NRTIs): i.e., zidovudine, didanosine, zalcitabine, stavudine, lamivudine, abacavir, emtricitabine and nucleotide reverse transcriptase inhibitors (NtRTIs) (i.e., tenofovir disoproxil fumarate); (ii) non-nucleoside reverse transcriptase inhibitors (NNRTIs): i.e., nevirapine, delavirdine, efavirenz, emivirine; and (iii) protease inhibitors (PIs): i.e., saquinavir, ritonavir, indinavir, nelfinavir, amprenavir, and lopinavir. In addition to the reverse transcriptase and protease reaction, various other events in the HIV replicative cycle can be considered as potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120

(polysulfates, polysulfonates, polycarboxylates, polyoxometalates, polynucleotides, and neg. charged albumins); (ii) viral entry, through blockade of the viral coreceptors CXCR4 (i.e., bicyclam (AMD3100) derivs.) and CCR5 (i.e., TAK-779 derivs.); (iii) virus-cell fusion, through binding to the viral envelope glycoprotein gp41 (T-20, T-1249); (iv) viral assembly and disassembly, through NCp7 zinc finger-targeted agents [2,2'dithiobisbenzamides (DIBAs), azadicarbonamide (ADA)]; (v) proviral DNA integration, through integrase inhibitors such as 4-aryl-2,4-dioxobutanoic acid derivs.; (vi) viral mRNA transcription, through inhibitors of the transcription (transactivation) process (flavopiridol, fluoroquinolones). Also, various new NRTIs, NNRTIs , and PIs have been developed that possess, resp.: (i) improved metabolic characteristics (i.e., phosphoramidate and cyclosaligenyl pronucleotides by-passing the first phosphorylation step of the NRTIs), (ii) increased activity ["second" or "third" generation NNRTIs (i.e., TMC-125, DPC-083)] against those HIV strains that are resistant to the "first" generation NNRTIs, or (iii), as in the case of PIS, a different, modified peptidic (i.e., azapeptidic (atazanavir)) or non-peptidic scaffold (i.e., cyclic urea (mozenavir), 4-hydroxy-2-pyrone (tipranavir)). Non-peptidic PIs may be expected to inhibit HIV mutant strains that have become resistant to peptidomimetic PIs.

REFERENCE COUNT: 187 THERE ARE 187 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:476090 CAPLUS

DOCUMENT NUMBER: 138:82710

TITLE: New developments in anti-HIV chemotherapy

AUTHOR(S): De Clercq, Erik

CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.

SOURCE: Biochimica et Biophysica Acta, Molecular Basis of Disease (2002), 1587(2-3), 258-275
CODEN: BBADEX; ISSN: 0925-4439

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Virtually all the compds. that are currently used, or are subject of advanced clin. trials, for the treatment of human immunodeficiency virus (HIV) infections, belong to one of the following classes: (i) nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs): i.e. zidovudine (AZT), didanosine (ddI), zalcitabine (ddC), stavudine (d4T), lamivudine (3TC), abacavir (ABC), emtricitabine [(-)FTC], tenofovir disoproxil fumarate; (ii) non-nucleoside reverse transcriptase inhibitors (NNRTIs): i.e. nevirapine, delavirdine, efavirenz, emivirine; and (iii) protease inhibitors (PIs): i.e. saquinavir, ritonavir, indinavir, nelfinavir, amprenavir and lopinavir. In addition to the reverse transcriptase (RT) and protease reaction, various other events in the HIV replicative cycle can be considered as potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120 (polysulfates, polysulfonates, polycarboxylates, polyoxometalates, polynucleotides, and neg. charged albumins); (ii) viral entry, through blockade of the viral coreceptors CXCR4 [bicyclam (AMD3100) derivs.] and CCR5 (TAK-779 derivs.); (iii) virus-cell fusion, through binding to the viral envelope glycoprotein gp41 (T-20, T-1249); (iv) viral assembly and disassembly, through NCp7 zinc finger-targeted agents [2,2'-dithiobisbenzamides (DIBAs), azadicarbonamide (ADA)]; (v) proviral DNA integration, through integrase inhibitors such as 4-aryl-2,4-dioxobutanoic acid derivs.; (vi) viral mRNA transcription,

through inhibitors of the transcription (transactivation) process (flavopiridol, fluoroquinolones). Also, various new NRTIs, NNRTIs and PIs have been developed that possess, resp.: (i) improved metabolic characteristics (i.e. phosphoramidate and cyclosaligenyl pronucleotides by-passing the first phosphorylation step of the NRTIs), (ii) increased activity ["second" or "third" generation NNRTIs (i.e. TMC-125, DPC-083)] against those HIV strains that are resistant to the "first" generation NNRTIs, or (iii) as in the case of PIs, a different, nonpeptidic scaffold [i.e. cyclic urea (mozenavir), 4-hydroxy-2-pyrone (tipranavir)]. Nonpeptidic PIs may be expected to inhibit HIV mutant strains that have become resistant to peptidomimetic PIs. Given the multitude of mol. targets with which anti-HIV agents can interact, one should be cautious in extrapolating the mode of action of these agents from cell-free enzymic assays to intact cells. Two examples in point are l-chicoric acid and the nonapeptoid CGP64222, which were initially described as an integrase inhibitor or Tat antagonist, resp., but later shown to primarily act as virus adsorption/entry inhibitors, the latter through blockade of CXCR4.

REFERENCE COUNT: 148 THERE ARE 148 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:323471 CAPLUS
DOCUMENT NUMBER: 137:210252
TITLE: Highlights in the development of new antiviral agents
AUTHOR(S): De Clercq, E.
CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.
SOURCE: Mini-Reviews in Medicinal Chemistry (2002), 2(2), 163-175
CODEN: MMCIAE; ISSN: 1389-5575
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The potential of a large variety of new compds. and new strategies for the treatment of virtually all major virus infections has been addressed. This includes, for the treatment of HIV infections, virus adsorption inhibitors (cosalane derivs., cyanovirin-N), co-receptor antagonists (TAK-779, AMD3100), viral fusion inhibitors (pentafuside T-20, betulinic acid derivs.), viral uncoating inhibitors (azodicarbonamide), nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs:emtricitabine, amdoxovir, dOTC, d4TMP prodrugs, tenofovir disoproxil fumarate), non-nucleoside reverse transcriptase inhibitors (NNRTIs: thiocarboxanilide UC-781, capravirine, SJ-3366, DPC 083, TMC 125/R165335), integrase inhibitors (diketo acids), transcription inhibitors (temacrazine, flavopiridol), protease inhibitors (atazanavir, mozenavir, tipranavir); for the treatment of RSV and paramyxovirus infections, viral fusion inhibitors (R170591, VP-14637, NMS03); for the treatment of picornavirus infections, viral uncoating inhibitors (pleconaril); for the treatment of pesti- (hepaci-, flavi-) virus infections, RNA replicase inhibitors (VP-32947); for the treatment of herpesvirus (HSV, VZV, CMV) infections, DNA polymerase inhibitors (A-5021, L- and D-cyclohexenylguanine).

REFERENCE COUNT: 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:849375 CAPLUS
DOCUMENT NUMBER: 136:128489
TITLE: New developments in anti-HIV chemotherapy

AUTHOR(S): De Clercq, Erik
CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.
SOURCE: Current Medicinal Chemistry (2001), 8(13), 1543-1572
CODEN: CMCHE7; ISSN: 0929-8673
PUBLISHER: Bentham Science Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Virtually all the compds. that are currently used, or under advanced clin. trial, for the treatment of HIV infections, belong to one of the following classes: (i) nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs): i.e., zidovudine (AZT), didanosine (ddI), zalcitabine (ddC), stavudine (d4T), lamivudine (3TC), abacavir (ABC), emtricitabine [(-)FTC], tenofovir (PMPA) disoproxil fumarate; (ii) non-nucleoside reverse transcriptase inhibitors (NNRTIs): i.e., nevirapine, delavirdine, efavirenz, emivirine (MKC-442); and (iii) protease inhibitors (PIs): i.e., saquinavir, ritonavir, indinavir, nelfinavir, amprenavir, and lopinavir. In addition to the reverse transcriptase and protease step, various other events in the HIV replicative cycle are potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120 (polysulfates, polysulfonates, polyoxometalates, zintevir, neg. charged albumins, cosalane analogs); (ii) viral entry, through blockade of the viral coreceptors CXCR4 and CCR5 [bicyclams (i.e. AMD3100), polyphemusins (T22), TAK-779, MIP-1α LD78β isoform]; (iii) virus-cell fusion, through binding to the viral glycoprotein gp41 [T-20 (DP-178), T-1249 (DP-107), siamycins, betulinic acid derivs.]; (iv) viral assembly and disassembly, through NCp7 zinc finger-targeted agents [2,2'-dithiobisbenzamides (DIBAs), azadicarbonamide (ADA) and NCp7 peptide mimics]; (v) proviral DNA integration, through integrase inhibitors such as L-chicoric acid and diketo acids (i.e. L-731,988); (vi) viral mRNA transcription, through inhibitors of the transcription (transactivation) process (fluoroquinolone K-12, Streptomyces product EM2487, temacrazine, CGP64222). Also, in recent years new NRTIs, NNRTIs and PIs have been developed that possess resp. improved metabolic characteristics (i.e. phosphoramidate and cyclosaligenyl pronucleotides of d4T), or increased activity against NNRTI-resistant HIV strains [second generation NNRTIs, such as capravirine and the novel quinoxaline, quinazolinone, phenylethylthiazolylthiourea (PETT) and emivirine (MKC-442) analogs], or, as in the case of PIs, a different, non-peptidic scaffold [i.e. cyclic urea (DMP 450), 4-hydroxy-2-pyrone (tipranavir)]. Given the multitude of mol. targets with which anti-HIV agents can interact, one should be cautious in extrapolating from cell-free enzymic assays to the mode of action of these agents in intact cells. A number of compds. (i.e. zintevir and L-chicoric acid, on the one hand; and CGP64222 on the other hand) have recently been found to interact with virus-cell binding and viral entry in contrast to their proposed modes of action targeted at the integrase and transactivation process, resp.

REFERENCE COUNT: 228 THERE ARE 228 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:516924 CAPLUS
DOCUMENT NUMBER: 135:282589
TITLE: New developments in anti-HIV chemotherapy
AUTHOR(S): De Clercq, Erik
CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.

SOURCE: Farmaco (2001), 56(1-2), 3-12
CODEN: FRMCE8; ISSN: 0014-827X
PUBLISHER: Elsevier Science S.A.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with refs. Virtually all the compds. that are currently used, or under advanced clin. trial, for the treatment of HIV infections, belong to one of the following classes: (i) nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs): i.e. zidovudine, didanosine, zalcitabine, stavudine, lamivudine, abacavir, emtricitabine, tenofovir (PMPA) disoproxil fumarate; (ii) non-nucleoside reverse transcriptase inhibitors (NNRTIs): i.e. nevirapine, delavirdine, efavirenz, emivirine; and (iii) protease inhibitors (PIs): i.e. saquinavir, ritonavir, indinavir, nelfinavir and amprenavir. In addition, various other events in the HIV replicative cycle are potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120; (ii) viral entry, through blockade of the viral coreceptors CXCR4 and CCR5; (iii) virus-cell fusion; (iv) viral assembly and disassembly; (v) proviral DNA integration; (vi) viral mRNA transcription. Also, new NRTIs, NNRTIs and PIs have been developed that possess resp. improved metabolic characteristics, or increased activity against NNRTI-resistant HIV strains or, as in the case of PIs, a different, non-peptidic scaffold. Given the multitude of mol. targets with which anti-HIV agents can interact, one should be cautious in extrapolating from cell-free enzymic assays to the mode of action of these agents in intact cells.
REFERENCE COUNT: 103 THERE ARE 103 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:303621 CAPLUS
DOCUMENT NUMBER: 135:70504
TITLE: New developments in anti-HIV chemotherapy
AUTHOR(S): De Clercq, Erik
CORPORATE SOURCE: Rega Institute for Medical Research, Katholieke Universiteit Leuven, Louvain, B-3000, Belg.
SOURCE: Pure and Applied Chemistry (2001), 73(1), 55-66
CODEN: PACHAS; ISSN: 0033-4545
PUBLISHER: International Union of Pure and Applied Chemistry
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 103 refs. Virtually all the compds. that are currently used, or under advanced clin. trial, for the treatment of HIV infections, belong to one of the following classes: (i) nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs): i.e., zidovudine, didanosine, zalcitabine, stavudine, lamivudine, abacavir, emtricitabine, tenofovir (PMPA), and disoproxil fumarate; (ii) non-nucleoside reverse transcriptase inhibitors (NNRTIs): i.e., nevirapine, delavirdine, efavirenz, and emivirine; and (iii) protease inhibitors (PIs): i.e., saquinavir, ritonavir, indinavir, nelfinavir, and amprenavir. In addition, various other events in the HIV replicative cycle are potential targets for chemotherapeutic intervention: (i) viral adsorption, through binding to the viral envelope glycoprotein gp120; (ii) viral entry, through blockade of the viral coreceptors CXCR4 and CCR5; (iii) virus-cell fusion; (iv) viral assembly and disassembly; (v) proviral DNA integration; and (vi) viral mRNA transcription. Also, new NRTIs, NNRTIs, and PIs have been developed that possess resp. improved metabolic characteristics, or increased activity against NNRTI-resistant HIV strains

or, as in the case of PIs, a different, nonpeptidic scaffold. Given the multitude of mol. targets with which anti-HIV agents can interact, one should be cautious in extrapolating from cell-free enzymic assays to the mode of action of these agents in intact cells.

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L1 19 S TMC278
L2 2 S L1 AND TENOFOVIR
L3 1232 S TENOFOVIR
L4 103 S L3 AND NNRTI
L5 42 S L4 AND EMTRICITABINE
L6 0 S L5 AND COMPOSITIONS
L7 41 S L5 AND HIV
L8 7 S L7 AND PY <2003